U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 05 July 2006

CASE NO.: 2003-BLA-5265

In the Matter of:

FREDDIE D. JARRELL
Claimant

v.

CONSOLIDATION COAL COMPANY
Employer

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party in Interest

DECISION AND ORDER ON REMAND – AWARDING BENEFITS

This case arises from a claim for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901 et seq. (Act). I conducted a hearing on June 23, 2004 in Beckley, West Virginia, and issued a Decision and Order – Awarding Benefits on December 2, 2004. (D. & O.). Employer appealed this decision to the Benefits Review Board (Board), which issued a Decision and Order on December 29, 2005. (Board D. & O.). The Board affirmed my finding that the claim was timely filed, as well as my findings pursuant to 20 C.F.R. §§ 718.202(a)(2)-(a)(3), 718.203(b), and 718.204(b) because they are unchallenged on appeal. (Board D. & O. at 2, nt 4). The Board also affirmed my finding that the x-ray evidence is sufficient to establish the existence of pneumoconiosis at § 718.202(a)(1).

The Board vacated a number of my findings related to the CT scan evidence. The Board vacated my finding regarding Dr. Younis's CT scan interpretation and instructed that I determine whether the fact that Dr. Younis's evaluation was not performed in connection with Claimant's claim for benefits affects its probative value. Specifically, I must consider if because the test was not performed in connection with this litigation, Dr. Younis may have had no reason to explicitly note that the CT scan showed no evidence of pneumoconiosis. The Board also vacated my finding that the CT scan evidence was accorded less weight, and instructed that I provide an explanation regarding my credibility determinations of Drs. Cohen and Zaldivar on the issue. Additionally, the Board vacated my ruling excluding Dr. Wiot's deposition testimony from the record, ordering that I consider whether it should be admitted pursuant to § 718.107(b).

The Board vacated my findings that Claimant established the existence of clinical and legal pneumoconiosis pursuant to §§ 718.201 and 718.202(a)(4), and instructed that I reconsider the relevant evidence. Specifically, the Board mandated that I: (1) reconsider Dr. Zaldivar's report and testimony, and provide a more detailed analysis of his findings; (2) consider Dr. Crisalli's deposition testimony in evaluating his findings; (3) consider the conflicting opinions of Drs. Zaldivar, Crisalli, and Koenig and provide an explanation regarding the weight given to each opinion; (4) determine whether my finding that Dr. Koenig's pulmonary function study is entitled to no weight at § 718.204(b)(2)(i) affects the credibility of Dr. Koenig's finding of legal pneumoconiosis; (5) consider whether Dr. Cohen's testimony regarding potential treatment for asthma in Claimant supports his conclusions regarding the existence of legal pneumoconiosis; (6) explain my reasoning behind my finding that the studies cited by Dr. Cohen supported his conclusion when I reexamine his opinion; (7) reconsider the opinions of Drs. Porterfield, Cohen, Koenig, Zaldivar, and Crisalli on the existence of clinical pneumoconiosis and render credibility determinations regarding this evidence, including whether Dr. Cohen's finding of clinical pneumoconiosis is adequately reasoned and documented in light of his testimony that there is not good evidence of the disease; and (8) reconsider all of the relevant evidence pursuant to § 718.202(a), addressing the significance that the negative CT scan interpretations may have on the x-ray evidence.

If I reach the issue of disability causation on remand, the Board instructed that I reconsider all of the relevant evidence regarding whether Claimant's total respiratory disability is due to pneumoconiosis, explaining the rationale for my conclusions. In reweighing the medical opinion evidence pursuant to § 718.204(c), I must consider the holdings of the Court of Appeals for the Fourth Circuit in *Scott v. Mason Coal Co*., 289 F.3d 263 (4th Cir. 2002); *Dehue Coal Co. v. Ballard*, 65 F.3d 1189 (4th Cir. 1995); *Hobbs v. Clinchfield Coal Co.*, 45 F.3d 819 (4th Cir. 1995); and *Toler v. Eastern Assoc. Coal Corp.*, 43 F.3d 109 (4th Cir. 1995). My finding regarding the date of entitlement was also vacated and is to be reconsidered, if reached, on remand.

Dr. Wiot's Deposition Testimony

Dr. Jerome F. Wiot was deposed on June 2, 2004. The admissibility of his deposition was one of the issues contested by the parties in their pre-hearing motions. On July 11, 2003, the Director submitted a brief in response to the parties pre-hearing motions. The Director noted that the "testimony of any witness or party may be taken by deposition… subject to the limitations on the scope of the testimony contained in § 725.457(d)." § 725.458. However, "[n]o person shall be permitted to testify at deposition or trial under § 725.458 unless that person meets the requirements of § 725.414(c)." § 725.457(c). Section 725.414(c) provides that a physician who prepared a report that is admitted in the record may testify with respect to the claim at a deposition or trial. It also stated that where a physician did not prepare a report, his testimony may be offered and construed as a medical report if the offering party has not exceeded the limitations provided by this section. (*Id.*). The Director noted that Dr. Wiot did not prepare a medical report. As such, Employer could only submit Dr. Wiot's deposition testimony into the record as one of the two medical opinions permitted by § 725.414(a)(3)(i) and (c), as the Employer had not made a showing of good cause pursuant to §725.456(b)(1).

On June 14, 2004, the undersigned excluded Dr. Wiot's deposition by pre-trial order. At the hearing held on June 23, 2004, Employer proffered Dr. Wiot's deposition for admission into evidence pursuant to § 718.107(b) but the undersigned deferred to the Director's position and excluded the deposition from the record. (TR 19-21; 40-41). The Board instructed that I consider on remand whether Dr. Wiot's deposition should be admitted pursuant to § 718.107(b).

Each party submitted an extensive brief on remand. Employer argues that the deposition of Dr. Wiot should be considered as relevant evidence and permissible testimony concerning the value of "other medical evidence." (Employer's Brief on Remand at 5-6). At the hearing, Employer argued that the CT scan evidence is relevant to refuting the existence of pneumoconiosis in Claimant, and that §718.107(b) dictates that the party submitting the "other evidence" bears the burden of establishing its relevancy. (*Id.*; TR at 20; 40-41). Claimant argues that Dr. Wiot's testimony establishes the uncontested point that CT scans are an accepted medical procedure that are relevant to establishing or refuting a claimant's entitlement to benefits, but that the testimony of the other physicians whom he deposed on the issue is sufficient. (Claimant's Mem. of Law on Remand at 5; 7). Claimant also argues that §718.107(b) is silent on the means by which a party may fulfill its burden of proving that "other medical evidence" is medically acceptable and relevant, and that deposition testimony is not the most cost effective method of establishing an issue. (*Id.*).

Subsequent to the Board's remand of the instant matter, it considered and ruled on the admissibility of deposition testimony for the purposes of § 718.107(b). In *Webber v. Peabody Coal Co*., 23 BLR - __, BRB No. 05-0335 BLA (Jan. 27, 2006) (en banc), the Board held that deposition testimony pertaining to the medical acceptability and relevance of "other medical evidence," such as CT scans, is admissible under § 718.107(b). However, the Board noted that where a physician's statement or testimony offered to satisfy the party's burden of proof at § 718.107(b) contains discussion of the miner or his condition, that testimony may be severed or excluded subject to § 725.414. In light of the foregoing, selected portions of Dr. Wiot's deposition attesting to the medical acceptability and relevance of the CT scan evidence are admitted into evidence.[1]

Dr. Younis's CT Scan Interpretation

The Board instructed that I determine whether the fact that Dr. Younis's CT scan was not performed in connection with Claimant's claim for benefits affects its probative value, and whether because the test was not performed in connection with this litigation, Dr. Younis may have had no reason to explicitly note that the CT scan showed no evidence of pneumoconiosis. I accorded Dr. Younis's interpretation no weight because it was silent as to the existence of pneumoconiosis. (D. & O. at 8).

Dr. Younis's CT scan interpretation was conducted for diagnostic purposes in connection with Claimant's treatment. Employer argues that Dr. Younis's interpretation should be deemed a negative interpretation. It points out that clinical treatment is usually for the purpose of identifying what diseases are present. Claimant agrees, offering that the most "intellectually

[1] The admissible portions of Dr. Wiot's deposition include: p. 11, line 4 through p. 12, line 22; p. 14, lines 16 – 20; p. 15, line 12 through p. 18, line 6; p. 20, line 13 through p. 23, line 10; p. 26, line 12 through p. 28, line 21.

honest" course is to assume that Dr. Younis's silence meant that he looked for, but did not see, pneumoconiosis.

In *Marra v. Consolidation Coal Co.*, 7 BLR 1-216 (1984), the Board determined that it was "unreasonable to adopt a standard that requires a physician to report not only the presence of abnormalities but also the absence of disease process." The Board held that an ALJ may "generally assume" that if the physician reading the x-ray does not mention pneumoconiosis, then pneumoconiosis is not present." In light of the foregoing, I find that Dr. Younis's basis for interpreting the CT scan does not affect its probative value and thus, the test will be accorded equal weight to the interpretations of the other physicians. Further, I find that Dr. Younis's CT scan interpretation shall be deemed an interpretation negative for pneumoconiosis.

Weight of the CT Scan Evidence

The Board vacated my finding that the CT scan evidence was accorded less weight and instructed that I provide an explanation regarding my credibility determinations as to the testimony of Drs. Cohen and Zaldivar on the issue. Additionally, I will consider the newly admitted deposition testimony of Dr. Wiot and the CT scan interpretation of Dr. Younis in making my determination on this point.

All four CT scan interpretations are negative for clinical pneumoconiosis. However, Drs. Wiot, Shipley, and Younis noted the presence of bullous emphysematous changes in the lungs, and Dr. Ahmed stated that the CT scan findings suggested COPD. Dr. Cohen testified that the CT scan is "probably the best test to evaluate the lung for emphysema other than pathology." (CX 14 at 28). Dr. Cohen also suggested that the CT scan was a standard CT, and not done "in the technique or protocol [used] to look for small nodules or interstitial lung disease." (*Id.* at 68). He testified that the standard ten millimeter slices were blurrier and coarser than many routine CT scans cut at 5 millimeters. (*Id.* at 69). He stated that the CT scan wasn't that helpful in determining the existence of pneumoconiosis because it was not a high resolution CT scan. (*Id.* at 88). Dr. Cohen stated that he was not certain how superior ten millimeter cut CT scans are to plain chest radiography, but that "in the pleural, better than a plain x-ray." (*Id.* at 89).

Dr. Zaldivar testified that CT scans are helpful in diagnosing the existence of pneumoconiosis in that they capture a smaller portion of the lung. (EX 19 at 45-46). He stated that a conventional CT scan is ten millimeters in thickness compared to the entire thickness of the chest on x-ray. (*Id.* at 46). Dr. Zaldivar observed that Claimant's CT scan was done at ten millimeters, though recently some CT scans are cut at seven millimeters. (*Id.*). Dr. Wiot testified that CT scans provide a better look at the lungs than chest x-rays because they are a dissection. (Wiot Depo. at 11-12). Dr. Wiot testified that Claimant's CT scan was a standard ten millimeter cut, but that seven millimeter cuts are done more often because of newer equipment. (*Id.* at 16). He stated that CT scans are generally more sensitive than x-rays. (*Id.* at 17). He added, however, that CT scans and x-rays are complimentary procedures because despite the fact that the CT scan is more sensitive in most cases, he has occasionally seen x-ray findings compatible with simple coal workers' pneumoconiosis that he could not appreciate on a CT scan. (*Id.* at 17). Dr. Wiot also noted that the presence of emphysema is not a radiographic finding, but rather a pulmonary function finding, such that it is able to be read, but determining the etiology is left to the

clinicians. (*Id.* at 22-23). In response to a question about whether ten millimeter cuts are blurrier, Dr. Wiot stated that sometimes nodules are visible on a ten millimeter cut and not on a one millimeter cut because a one millimeter cut might not be cut at the right level. (*Id.* at 28).

The physician testimony universally establishes that CT scan evidence is relevant and useful as a diagnostic tool. The Court of Appeals for the Seventh Circuit has rejected the argument that negative CT scan readings are conclusive of the absence of pneumoconiosis because they are the most sophisticated diagnostic tool available. *Consolidation Coal v. Director, OWCP*, 294 F.3d 885 (7th Cir. 2002). Citing to comments underlying the amended regulations, the court noted that the Department has rejected the view that a CT scan alone "is sufficiently reliable that a negative result effectively rules of the existence of pneumoconiosis." *Id*. at 890-91; 65 Fed. Reg. 79, 920, 79, 945 (Dec. 20, 2000). Thus, the CT scan has not been interpreted as the "best" evidence for establishing or refuting the existence of pneumoconiosis. The physician testimony supports this conclusion as well.

Currently there no standards in place governing the administration of CT scans (as there are with the ILO classification system for x-rays), nor a test by which one may assess a reviewing physician's qualifications. Additionally, while I find that Claimant's ten millimeter cut CT scan was reliable, each physician noted that seven millimeter cuts are also performed. Thus, the physician opinion evidence demonstrates that the technology is improving and higher resolution CT scans are available. Further, Dr. Wiot's testimony regarding higher resolution scans does not address Dr. Cohen's assertion that clarity improves with a higher resolution. Therefore, the evidence indicates that a ten millimeter scan is rendered less than optimal for assessing smaller nodules of a lesser profusion apparent in Claimant's x-rays. As a result of the foregoing, I find that the CT scan evidence is accorded diminished weight in establishing the existence of clinical pneumoconiosis.

Clinical Pneumoconiosis

The Board affirmed my findings that the x-ray evidence is sufficient to establish the existence of pneumoconiosis at § 718.202(a)(1), that there is no biopsy evidence and that the enumerated presumptions are not applicable to this claim. § 718.202(a)(2)-(a)(3).

The Board vacated my finding that Claimant established the existence of clinical pneumoconiosis pursuant to §§ 718.201 and 718.202(a)(4). The Board instructed that I reconsider the opinions of Drs. Porterfield, Cohen, Koenig, Zaldivar, and Crisalli on the existence of clinical pneumoconiosis and render credibility determinations regarding this evidence, including whether Dr. Cohen's finding of clinical pneumoconiosis is adequately reasoned and documented in light of his testimony. I must also I consider all of the relevant evidence pursuant to § 718.202(a), addressing the significance that the negative CT scan interpretations may have on the x-ray evidence. The record contains the medical opinions of five physicians and four interpretations of CT scan evidence.

Dr. Porterfield based his finding of clinical pneumoconiosis on an 11/15/01 positive x-ray and coal dust exposure. (DX 12). Dr. Crisalli based his determination that Claimant does not have clinical pneumoconiosis upon his review of the x-ray evidence, which included Dr. Willis's

positive x-ray interpretation not in the record, Dr. Wheeler's x-ray interpretation of no pneumoconiosis, Dr. Patel's positive x-ray interpretation, and Dr. Zaldivar's x-ray interpretation of no pneumoconiosis. (DX 25; EX 10; EX 21 at 50-53). Dr. Zaldivar based his determination that Claimant does not have clinical pneumoconiosis upon his review of the x-ray evidence, which included his own x-ray interpretation, Dr. Wheeler's x-ray interpretation of no pneumoconiosis, Dr. Patel's positive x-ray interpretation, Dr. Ahmed's positive x-ray interpretation, and Dr. Wiot's x-ray interpretation of no pneumoconiosis. (EX 4; EX 19 at 39-45). Dr. Koenig reviewed the positive x-ray interpretation of Dr. Patel, the negative x-ray interpretations of Drs. Zaldivar and Wiot, and the x-ray interpretation of Dr. Binns, not of record. He concluded that there was an absence of definitive x-ray evidence demonstrating pneumoconiosis. (CX 4).

Dr. Cohen reviewed the positive x-ray interpretations of Drs. Patel, Miller, Ahmed, and Capiello, and the negative x-ray interpretations of Drs. Wheeler and Wiot. (CX 4; CX 14 at 24-28, 64-66, 87-90). When asked if Claimant has clinical pneumoconiosis, Dr. Cohen testified, "I think he does... I think that he probably does but I don't have good evidence." (CX 14 at 88). Dr. Cohen testified that the x-ray evidence in the record supports both the existence and absence of pneumoconiosis. (*Id*. at 24-25). He also stated that one could have a negative chest x-rays and still have interstitial lung disease. (*Id.* at 27). He added that the CT scan was not helpful, as it might have been if it were conducted at a higher resolution. (*Id.* at 88). He further testified that Claimant "probably does have medical pneumoconiosis. The medical evidence for that is the x-rays. The x-rays have been read positive and negative, but I certainly, to a reasonable degree of medical certainty, see that he has lung function impairment, which is due to, I think, his thirty-four years of coal mine dust exposure." (*Id.* at 136).

I find that Dr. Cohen's discussion of evidence contrary to his position demonstrates that his opinion is comprehensive, adequately reasoned, and adequately documented. It also demonstrates that his opinion is not based exclusively on Claimant's history of coal dust exposure, as Employer suggests. Additionally, Dr. Cohen reviewed more x-ray interpretations than any other physician in this case. However, despite Dr. Cohen's thoughtful deliberation of conflicting evidence, the conclusion he draws therefrom amounts to an equivocation, and therefore, I accord his opinion diminished weight. (*Griffith v. Director, OWCP*, 49 F.3d 184 (6th Cir. 1995) (Treating physician's opinion entitled to little weight where he concluded that the miner "probably had black lung disease.")

The weight to be accorded physicians' opinions rests with the trier-of-fact. *Bogan v. Consolidation Coal Co*., 6 BLR 1-1000 (1984). I choose to accord greater weight to the opinion of Dr. Porterfield over the opinions of Drs. Koenig, Crisalli, Cohen, and Zaldivar because Drs. Koenig, Crisalli, and Zaldivar determined that the x-ray evidence did not support the existence of pneumoconiosis, contrary to my finding, and Dr. Cohen's conclusion is equivocal. *Island Creek Coal v. Compton*, 211 F.3d 203 (4th Cir. 2000) (The court determined that the ALJ erred in crediting Dr. Gaziano's opinion that the claimant had coal workers' pneumoconiosis based solely on an x-ray taken in conjunction with his examination, contrary to the ALJ's determination that the x-ray evidence did not establish pneumoconiosis.); *See also Consolidation Coal v. Swiger*, 98 Fed. Appx. 227, 236 (4th Cir, 2004), citing *Sterling Smokeless Coal Co. v. Akers*, 131 F.3d 438, 441 (4th Cir. 1997) (The *Swiger* court reasoned that the ALJ could

rightfully discount medical opinions that contradicted his findings where he determined that the x-ray evidence was inconclusive rather than negative, as the medical opinions concluded.).

However, I also find that the physician opinion evidence as a whole shall be accorded diminished weight, as each of the physicians based their assessments on an incomplete review of the x-ray evidence of record. The Board has held that an administrative law judge (ALJ) "may legitimately assign less weight to a medical opinion which presents an incomplete picture of the miner's health." *Stark v. Director, OWCP*, 9 BLR 1-36 (1986), citing *Rickey v. Director, OWCP,* 7 BLR 1-106 (1984). Not one of the five physicians reviewed all of the x-ray interpretations of record. Dr. Zaldivar testified to the importance of an empirical review:

> Counsel: Doctor, from a medical standpoint, is it better to have more x-ray interpretations or less x-ray interpretations?
> Dr. Zaldivar: Well, one wants to have as much information as possible, which include [sic] x-ray interpretation, by individuals whose readings one trusts because they are well-known in the field or because they have extensive experience. But the more information available, the better, regardless of whether it is breathing tests, chest x-rays or whatever.
> Counsel: If a person looking at Mr. Jarrell's health only considered a limited number of x-rays, might they get the wrong idea?
> Dr. Zaldivar: They might, yes.

(EX 19 at 45).

In summary, I determined, and the Board affirmed, my finding that the x-ray evidence is sufficient to establish the existence of pneumoconiosis. I determined that the CT scan evidence was completely negative for pneumoconiosis, but that it was of diminished import due to the level of the CT scan's resolution. Additionally, CT scans and x-rays are different diagnostic tests, both of which are relevant to establishing the existence of absence of pneumoconiosis, but neither of which can be said to be more probative than the other. Further, I reasoned that three physicians' opinions could reasonably be discounted as they were contrary to my findings, and that one opinion was equivocal. Finally, I determined that the physician opinion evidence as a whole was of diminished import because none of the five physicians reviewed all of the x-ray evidence of record. As such, I find that the evidence expounded above establishes that Claimant has clinical pneumoconiosis.

The June 18, 2003 Pulmonary Function Test

The Board instructed that I determine whether my finding that Dr. Koenig's pulmonary function study is entitled to no weight at § 718.204(b)(2)(i) affects the credibility of Dr. Koenig and his finding of legal pneumoconiosis.

It is error to discredit a physician's report solely because of his or her reliance upon non-qualifying tests where the physician also relied upon a physical examination, employment and medical histories, and the symptomatology of the miner. *Baize v. Director, OWCP*, 6 BLR 1-730 (1984); *Wike v. Bethlehem Mines Corp*., 7 BLR 1-593 (1984); *Coen v. Director, OWCP*, 7 BLR

1-30 (1984); *Sabett v. Director, OWCP*, 7 BLR 1-299 (1984). I reasoned that the June 18, 2003 pulmonary function study would be given no weight because it failed to provide an adequate number of tracings as required by the regulations. § 718.103. An ALJ should determine whether the deficiency in the study is essential to the reliability and probative value of the physician's report. *Dillon v. Peabody Coal. Co*., 11 BLR 1-113 (1988).

Dr. Koenig's report expressly stated that in addition to the June 18, 2003 test, he based his medical opinion on Claimant's history and physical examination, as well as on the x-ray interpretations of various doctors noted above, the medical reports of Drs. Zaldivar, Crisalli, and Porterfield, and the arterial blood gas studies and pulmonary function studies of the same. (CX 4). Dr. Koenig's discussion of the objective findings accounts for the results of each test separately and his report indicates that he was not exclusively, or even predominantly reliant on the results of his own pulmonary function study. (*Id*.). Therefore, neither Dr. Koenig's credibility nor the integrity of his findings is undermined by his review and consideration of the study at issue. However, any reliance or discussion of this study by Dr. Koenig and the other physicians of record shall be discounted when making my determination.

Legal Pneumoconiosis

The Board vacated my finding that Claimant established the existence of legal pneumoconiosis pursuant to §§ 718.201 and 718.202(a)(4). The Board instructed that I reconsider the reports and testimony of Drs. Zaldivar and Crisalli and provide a more detailed analysis of their findings, that I reconsider Dr. Cohen's testimony regarding potential treatment of Claimant for asthma and whether his reliance on the medical literature support his conclusions, and that I provide an explanation regarding the weight given to each doctor's opinion.

Dr. Porterfield

Dr. Porterfield diagnosed the miner with pneumoconiosis based on a positive x-ray, and chronic obstructive pulmonary disease (COPD) based on the miner's pulmonary function studies. He listed the etiology of these diseases as smoking and coal dust exposure. Because it is unclear from Dr. Porterfield's report whether both etiologies underlie his diagnosis of COPD, I find that Dr. Porterfield did not diagnose the miner with legal pneumoconiosis, and his report is therefore not probative on the issue.

Dr. Koenig

Dr. Koenig is board certified in pulmonary disease. Dr. Koenig is an associate professor of medicine at the University of Virginia School of Medicine. He is the Director of the Occupational Lung Disease Program, the Pulmonary Rehabilitation Program, and the Co-director of the Asthma Clinic and Research Center at the school. He is also a Pulmonary and Critical Care Consultant at the Salem Veterans Administration Medical Center. (CX 4). Dr. Koenig diagnosed legal pneumoconiosis in the form of COPD caused by coal dust exposure. He stated that COPD is inclusive of afflictions such as chronic bronchitis, emphysema, and asthma. (*Id*. at 4). Dr. Koenig found that the bullae on Claimant's chest x-ray, his low DLCO and elevated TLC,

his increased RV/TLC, and the incomplete reversibility of airflow obstruction with bronchodilator favor a diagnosis of COPD, including chronic bronchitis and emphysema, and excluding asthma. (CX 4 at 4). He stated that the airflow obstruction caused by asthma is completely reversible, and that DLCOs and TLCs are normal, citing one study in support of this proposition. (*Id.*). He stated that asthma is not associated with bullae formation. He explained that Claimant's intermittent improvement post-bronchodilator is not definitive evidence of asthma as COPD patients also experience improvement after such trials, citing three studies in support of this proposition. (*Id.*).

Dr. Koenig also observed that Claimant's smoking history was not of sufficient intensity or duration to cause clinically important COPD. Thus, he concluded that the only logical explanation for Claimant's COPD is coal dust exposure. Dr. Koenig cited to various medical literature in support of his observation that coal dust exposure can cause or contribute to clinically significant COPD. Additionally, Dr. Koenig acknowledged the existence of medical literature that concludes to the contrary, and cited two additional articles in an effort to reconcile the opposing conclusions. (*Id.* at 5).

Dr. Cohen

Dr. Cohen is board certified in internal medicine and a pulmonary specialist. In his deposition testimony, Dr. Cohen testified that he is a senior attending physician at the John H. Stroger, Jr., Hospital of Cook County. (CX 14 at 4). He is also associate professor at Rush University and assistant professor of medicine at the University of Illinois School of Public Health, Division of Environmental Occupational Health Sciences. (*Id.* at 4-5). He is the medical director of the Black Lung Clinics Program. Dr. Cohen diagnosed COPD in Claimant, caused in part by coal dust exposure. In his report, Dr. Cohen concluded that Claimant has a moderate to severe obstructive defect and a mild to moderate diffusion impairment which are resultant primarily from his thirty-four years of coal mine dust exposure, but also, to a lesser degree, his modest tobacco smoke exposure. (CX 1 at 11). Dr. Cohen cited a study that states that one pack year of smoking causes a similar degree of impairment for one year of underground exposure to coal dust. (*Id*. at 7). Dr. Cohen cited extensively to the medical literature in support of his contention that occupational coal dust exposure causes obstructive lung disease. (CX 1). He cited one study which stated that coal dust and tobacco smoke produce similar decrements in lung function. (*Id.* at 9).

Dr. Cohen disagreed with Drs. Crisalli and Zaldivar that Claimant has asthma. He stated that Claimant has COPD with a reversible component. (*Id.*). He stated that if Claimant had asthma, his FEV1 would reverse to normal, which it did not. He also stated that Claimant's most improved FEV1 was only 57%, which is a level of improvement not greater than moderate impairment. (*Id.*). Dr. Cohen reasoned that Claimant's symptoms are not diagnostic of bronchial asthma, and that he had no history of bronchospasm. (*Id.*). He cited a study which states that "asthma is often reversible either spontaneously or with treatment." (*Id.* at 10). He also cited a study that found that obstructive lung disease due to coal dust demonstrates bronchial hyper-responsiveness and reversibility. (*Id.*).

Dr. Cohen's deposition testimony reaffirms his conclusions. (CX 14). He testified that the diagnosis of asthma is a clinical diagnosis based on a patient's entire medical history and any history of other exposures and diseases. (*Id.* at 38). Dr. Cohen concluded that based on Claimant's clinical history, the presence of a diffusion impairment not seen in asthmatics, and the relatively small, varied response to bronchodilators, Claimant does not have asthma. (*Id*. at 38-39). Dr. Cohen acknowledged that remodeling can occur in the airways of asthmatics. (*Id.* at 95). He ruled out remodeling of the airways due to asthma because Claimant "never had a diagnosis of asthma, was never treated long term for asthma, and developed symptoms only after more than 20 years of exposure to dust." (*Id*. at 96, 131). Additionally, he stated that while adult onset asthma develops, it is usually in the twenties or thirties, and he would have developed it later in life. (*Id*. at 97).

Dr. Cohen testified that coal mine dust can cause emphysema, which is quite similar to that which is caused by tobacco smoke exposure. (CX 14 at 10). He testified that when assessing the impact of a claimant's smoking history and his exposure to coal mine dust, the cumulative lifetime exposure to coal dust and the dust control regulations in effect must be examined and compared to the claimant's smoking history. (*Id.* at 17-19). Dr. Cohen demonstrated knowledge of Claimant's various coal mine employment duties, and the variability of coal mine dust exposure depending on location and type of job. (CX 1 at 1; CX 14 at 21;128). He testified that he did not know if Claimant utilized breathing protection equipment, but stated that he relied on the length of coal mine employment as a surrogate to assess exposure.

He estimated that a significant smoking history begins at about twenty years, and perhaps ten years in very sensitive lungs. (*Id.* at 20). Dr. Cohen concluded, therefore, that Claimant's 7 ½ pack year history could not be discounted completely from having contributory effects on his pulmonary disease, but that the smoking history was small. (*Id.* at 20-21). By contrast, approximately fourteen years of Claimant's coal mine employment was prior to the implementation of dust control regulations. (*Id.* at 21).

Dr. Cohen testified that coal mine dust can cause bullae, which is associated with forms of emphysema, such as that caused by coal mine dust or smoking. (*Id.* at 22). He stated that clinical pneumoconiosis can also contribute to bullae formation. (*Id.*). Dr. Cohen testified that evidence of Claimant's emphysema was well-demonstrated on CT scan, upon which he relied in diagnosing emphysema. (*Id.* at 27-28). He also relied on Claimant's pulmonary function studies, which evidence moderate to severe obstructive lung disease as well as a mild diffusion impairment, usually indicative of emphysema. Dr. Cohen testified that Claimant's smoking history does not account for his pulmonary impairment. (*Id.* at 30-32).

Dr. Zaldivar

Dr. Zaldivar is board certified in pulmonary medicine. He is a clinical professor of medicine at West Virginia University School of Medicine as well as the Medical Osteopathic School of Medicine. (EX 19 at 51-52). He is also the Director of the Respiratory Therapy Department at Charleston Area Medical Center. (*Id.*). Dr. Zaldivar stated the that Claimant's pulmonary impairment is primarily due to asthma, due to the reversible nature of the obstruction by breathing test and normal breathing capacity, demonstrating that lung obstruction is due to

inflammation of the airways and not lung destruction. (EX 4). He stated that Claimant's obstruction looked like asthma as opposed to emphysema because his diffusing capacity was preserved and there was an improvement post-bronchodilator of over 12% accepted by the American Thoracic Society. (*Id.* at 21). He stated that radiologically, there is no evidence of coal workers' pneumoconiosis, and therefore pneumoconiosis is not contributing to airway damage. (EX 4 at 3).

In addition to asthma, Dr. Zaldivar stated that Claimant's pulmonary impairment is also likely due to emphysema from smoking. He stated that Claimant's asthma masks any emphysema that might be present on his breathing tests. (*Id*.). Additionally, he stated that the "physiological abnormality in this case is of emphysema and not that of damage of the airways caused by destruction nor damage by dust." (*Id*.). Dr. Zaldivar testified that Claimant's smoking history was significant because twenty pack years is the place where pulmonary problems are expected to develop, but that some individuals develop pulmonary problems even with a lesser history. (EX 19 at 11-12). He acknowledged that individuals exposed to coal mine dust develop pulmonary obstruction. (*Id.* at 32).

Dr. Zaldivar does not believe that coal dust exposure contributed to Claimant's disability. (*Id*. at 57). He stated that coal mine dust does not cause bullae or emphysema, it causes COPD, and that there is no physiological way to distinguish the two. (*Id* at 79.). He stated that since the CT showed bullae, that is evidence of emphysema from smoking because coal workers' pneumoconiosis does not cause bullae.

Dr. Zaldivar stated that Claimant's smoking habit is sufficient to have an effect on the miner in terms of aggravating his asthma, but that Claimant did not develop severe emphysema from smoking. (*Id.* at 37-38). Dr. Zaldivar testified that he does not believe Claimant's emphysema is solely attributable to smoking, but that Claimant has remodeled lungs, which is the combined result of asthma aggravated by smoking. (*Id.* at 79-80). Dr. Zaldivar testified that smoking's effect on asthma, which inflames the airways, produces repeated attacks and eventually results in a remodeling the lung, or emphysema. (*Id*. at 14). He noted that asthma is not completely reversible. (*Id.* at 33-34). He stated that in Claimant's case, "we are not dealing here with emphysema. We are dealing here with asthma, which is a different disease which is exacerbated by smoking at all." (*Id*. at 39).

Dr. Zaldivar stated that an individual may have more than one disease simultaneously. (*Id.* at 22). He stated that it is difficult to distinguish between asthma and emphysema based solely on reversibility and that diffusing capacity was also relevant. (*Id.* at 76). Dr. Zaldivar testified that there was not enough improvement, or reversibility, post-bronchodilator in either Dr. Porterfield's or Dr. Crisalli's pulmonary function tests to qualify for asthma. (*Id.* 23-25; 58-61). Dr. Zaldivar stated that bronchodilators do not help an individual with coal workers' pneumoconiosis because the problem is not one of inflammation that could be reversed by bronchodilators. (*Id.* 26-27). Likewise, he testified that people with emphysema do not respond to bronchodilators because the airway is destroyed, and that reversibility of air flow obstruction is not seen in patients with emphysema. (*Id.* at 73-74). Dr. Zaldivar contended that the conclusions of an authoritative occupational lung disease textbook, asserting that patients with emphysema often respond to bronchodilators, is incorrect. (*Id*. at 75).

Dr. Crisalli

Dr. Crisalli is board certified in pulmonary disease. Dr. Crisalli is an associate professor at West Virginia University School of Medicine. (DX 25). He is also the Chief of the Pulmonary Subsection at the Charleston Area Medical Center. (*Id*.). He is the Medical Director of the Medical Intensive Care Units there as well. (EX 21). Dr. Crisalli stated in his medical report that there is not sufficient evidence to support a diagnosis of coal workers' pneumoconiosis or any chronic dust disease of the lung caused by, significantly related to, or substantially aggravated by coal mine employment. (DX 25). He stated that the reversible component of Claimant's obstruction to airflow indicates asthma, and that his moderately severe degree of air-trapping supports a diagnosis of emphysema. (*Id*.; EX 21). He stated that neither of these conditions is related to coal dust exposure. (DX 25). He indicated that testing should be performed to determine if Claimant has a hereditary predisposition toward emphysema. (*Id.*). Dr. Crisalli's supplemental report reaffirms his position. (EX 10).

Dr. Crisalli testified that Claimant's significant improvement post-bronchodilator correlates with Claimant's history of wheezing which results in a diagnosis of asthma. (EX 21 at 27). Additionally, he found that an absence of family history for asthma does not preclude it from occurring in Claimant. (*Id.* at 88). He stated that complete reversibility in asthmatics is rare. (*Id.* at 28). He also established that Dr. Cohen incorrectly interpreted that asthma is "fully" reversible according to the study he quoted. (*Id*. at 44). Dr. Crisalli went on to note that reversibility is considered significant when indicated by an increase of 12% or greater in FEV1 post-bronchodilator, according to the American Thoracic Society. (*Id*. at 44-45). He determined that Claimant's mild diffusion defect is consistent with a diagnosis of asthma and perhaps also effected by the large degree of air-trapping. (*Id.* at 34-35). He added that Claimant's lung volumes showed a marked degree of air-trapping, which does not really occur with pneumoconiosis as it does with other diseases, and that the diffusion capacity was near normal. (*Id*. at 52.). He testified that asthma is a disease of variable obstruction, but that coal mine dust induced disease or coal workers' pneumoconiosis can be either static or progressive. (*Id*. at 40-41). He stated that Claimant's variable degree of obstruction as between his test and that of Dr Porterfield is supportive of his diagnosis of asthma. (*Id.* at 82).

Dr. Crisalli stated that the medical literature associates an obstruction of airflow with coal dust exposure. (*Id*. at 63-64). Dr. Crisalli stated that coal dust does not cause or aggravate asthma. (*Id.* at 52). He also stated that if Claimant had coal workers' pneumoconiosis, he would still diagnose asthma, and that Claimant's impairment is not related to the pneumoconiosis because of the reversibility on the spirometry. (*Id*. at 52). He testified that his concern with Claimant was his air-trapping and emphysema. He testified that there are different types of emphysema, such as hereditary, related to smoking, and related to coal dust. (*Id.* at 62).

He stated that Alpha-1 antitrypsin deficiency predisposes a person to develop emphysema, and occurs in about one out of three thousand people. He did not test Claimant for this condition. (*Id.* at 69). Dr. Crisalli was unsure of Claimant's family history for the disease, but he testified that a lack of history does not rule out the possibility of disease. (*Id.* at 72-73). He suggested that Claimant get a blood test for hereditary emphysema because he has a fair amount of emphysema and has bullous disease, but was not a long-term smoker. (*Id.* at 53; 67-

68). Dr. Crisalli stated that he has no other explanation for the emphysema, as Claimant's smoking history is mild and probably not the cause of the disease. (*Id*. at 53).

He stated that the smoking history would not be associated with the development of lung disease unless there is a hereditary predisposition to developing emphysema. (*Id.* at 60). He stated that air-trapping can be related to asthma as well as emphysema with bullae. (*Id.* at 58). He testified that asthma generally does not create bullae, and that it does not cause a reduced diffusion capacity until there is a restructuring of the lung. (*Id.* at 77-78). However, he stated that coal dust never causes such severe air-trapping as seen in Claimant, and is never associated with bullae. (*Id*. at 62). Claimant's air-trapping was significant but has varied somewhat, which he stated is consistent with asthma. (*Id.* at 92). He stated that if Claimant's emphysema was responsible for air-trapping he would expect static or progressive permanent changes. (*Id.* at 90). He also stated that the pulmonary function data may all be related to asthma and have nothing to do with emphysema. (*Id.* at 69).

Dr. Crisalli opined that emphysema related to coal dust is a microscopic diagnosis that would not manifest itself with these types of pulmonary functions. (*Id.*). He stated that emphysema detectable by pulmonary function exam or bullous disease is unrelated to coal mine dust. (*Id*. at 63). He stated that he did not diagnose coal workers' pneumoconiosis in Claimant, he diagnosed emphysema, but that Claimant's emphysema was not related to coal mine dust. (*Id.*). He stated that applying the medical literature to Claimant's case does not result in a diagnosis of a coal dust related disease. (*Id.* at 65).

Four physicians rendered opinions addressing the existence of legal pneumoconiosis in Claimant. Drs. Koenig and Cohen diagnosed Claimant with legal pneumoconiosis in the form of COPD; Drs. Zaldivar and Crisalli diagnosed Claimant with asthma and emphysema related to asthma and smoking. The physician opinion evidence indicates that Claimant's smoking history was modest. Dr. Koenig determined that Claimant's smoking history was insufficient to impact his pulmonary condition. Dr. Crisalli stated that Claimant's smoking history was not significant unless he had hereditary emphysema. Dr. Cohen concluded that Claimant's smoking history was small, but could not be discounted in evaluating Claimant's pulmonary impairment. Dr. Zaldivar agreed that Claimant's smoking history was less than significant, but that it would impact his pulmonary condition.

Drs. Crisalli and Zaldivar made their diagnosis of asthma based on reversibility of Claimant's airflow post-bronchodilator, on the basis that coal mine dust is not improved by the administration of such therapy. Drs. Crisalli and Zaldivar diagnosed Claimant's emphysema as lung remodeling resulting from a combination of untreated asthma aggravated by smoking, and, in Dr. Crisalli's case, alternatively as a rare hereditary emphysematic condition for which he did not test Claimant.

Drs. Koenig and Cohen referred to various studies in support of the position that intermittent improvement post-bronchodilator is not definitive evidence of asthma, as COPD patients also experience improvement after such trials. Furthermore, Dr. Zaldivar testified that two of the three pulmonary function studies do not have reversibility so significant as to justify a diagnosis of asthma. Additionally, Claimant has no personal or family history of asthma, has not

been prescribed medication for asthma, and was not diagnosed with asthma until Dr. Crisalli did so in May, 2002. Claimant did not develop breathing problems until he was approximately forty-seven, a decade or two after adult-onset asthma typically occurs.

Dr. Cohen concluded that based on Claimant's clinical history, the presence of a diffusion impairment not seen in asthmatics, and the relatively small, varied response to bronchodilators, Claimant does not have asthma. Dr. Cohen ruled out lung remodeling because Claimant had no history of asthma or bronchospasm. Thus, the reasoning I applied to Dr. Younis's CT scan interpretation is applicable here – the absence of evidence in the record pertaining to any personal or family history of asthma indicates that Claimant's condition is not asthma. Dr. Cohen's testimony regarding treating Claimant with bronchodilators does not contradict his conclusion that Claimant does not suffer from asthma. He stated that he administers bronchodilators to all of his COPD patients. He would do so to obtain "whatever marginal improvement" was possible, in an effort to provide the a good evaluation.

In *Swiger*, *supra*. at 7, a claimant's pulmonary condition improved when he was given bronchodilator medication, but the residual impairment was still disabling. The physicians agreed that pneumoconiosis would not be susceptible to bronchodilator therapy. However, the fact that the claimant's residual impairment was disabling suggested that a combination of factors was causing his pulmonary condition. The court reasoned that the ALJ could rightfully conclude that "the presence of the residual fully disabling impairment suggested that coal mine dust was a contributing cause of Swiger's condition." *Swiger*, 98 Fed. Appx. at 237, citing *Underwood v. Elkay Mining, Inc*., 105 F.3d 946, 949 (4th Cir. 1997).

The physician opinion evidence in the instant case demonstrates that an asthmatic condition is variable and does not need to be completely reversible. It also demonstrates that coal mine dust can cause or contribute to COPD, and that patients with COPD can also demonstrate reversibility. Therefore, I find that the better supported medical evidence demonstrates that asthma is not present, and Claimant suffers from COPD induced by coal dust exposure. However, even if asthma was present, it does not exclude the presence of legal pneumoconiosis. Thus, I find that given Claimant's modest smoking history, smoking could not have caused the lung remodeling suggested by Drs. Crisalli and Zaldivar, such that his emphysema is attributable to the combined effect of smoking and asthma over that of Claimant's exposure to coal mine dust.

In *Bethenergy Mines, Inc. v. Director, OWCP*, 50 Fed. Appx. 578, 582-83 (4th Cir. 2002) (unpub.), the court of appeals held that it was proper for the ALJ to accord greater weight to Dr. Rasmussen, who offered the most extensive research to support his opinion. The court upheld the ALJ's reasoning that Dr. Rasmussen's opinion was more persuasive because he cited seven articles and six epidemiological studies to support his position. *Id*. at 582. In discounting the opinions of other physicians who were critical of Dr. Rasmussen, the ALJ observed that the other opinions lacked the "definitiveness to outweigh the better reasoned and better supported report of Dr. Rasmussen…," that disagreement with "the medical experts Dr. Rasmussen cited were in the most general of terms," and that no physician's opinion critiqued "any particular study or specific data behind a study." *Id*. Further, the ALJ found that while one doctor disputed the underlying data of studies cited by Dr. Rasmussen, he did not specify which of the studies

had evidentiary problems. Additionally, while that doctor identified a more recent study that purported to support his position, he did not "identify the study by title or author."

Bethenergy is instructive on the facts of the instant case. Drs. Zaldivar and Crisalli disagree with the content of the articles cited by Drs. Cohen and Koenig, and in some instances, assert that the studies are not germane to the Claimant's case. (EX 19 at 35-36; 50; 55-56; EX 21 at 43-47; 63-67). However, Drs. Zaldivar and Crisalli's challenge to the medical literature fails to provide more than conclusory disagreement with the studies cited by Drs. Cohen and Koenig, and appended either as an excerpt or in bibliography format. Overall, Dr. Zaldivar and Crisalli fail to identify, proffer, or summarize literature in support of their contrary position.[2] Additionally, I would note that it is not the province of the ALJ to render a medical determination. The ALJ is imbued with the power to render credibility determinations, from which he ascertains a claimant's diagnoses, and applies the law to those facts.

Accordingly, I find that the opinions of Drs. Cohen and Koenig are accorded greater weight than those of Drs. Zaldivar and Crisalli on the issue of legal pneumoconiosis because they not only cite extensively to the medical literature in support of their conclusions, but because the latter doctors did not refute the studies, or the data underlying the studies with sufficient specificity.

Additionally, Dr. Zaldivar's deposition testimony is so rife with contradictions that the probative value of his opinion is significantly diminished. In Dr. Zaldivar's report, he stated that the abnormality at issue in Claimant's case was emphysema, and not destruction of the airways. In his deposition testimony he stated that people with emphysema do not respond to bronchodilator therapy because the airway *is* destroyed. He testified that repeated asthma attacks instigated by smoking eventually remodels the lung, which manifests as emphysema. He further stated that if bullae are present then one knows one is dealing with emphysema due to smoking because coal workers' pneumoconiosis does not cause bullae. He believes this despite his conclusion that Claimant did not develop severe emphysema from smoking. Finally, after testifying to the inter-relationship between asthma, smoking, and emphysema in Claimant, Dr. Zaldivar testified that in Claimant's case, "we are not dealing here with emphysema. We are dealing here with asthma, which is a different disease which is exacerbated by smoking at all." Due to Dr. Zaldivar's equivocal and contradictory testimony, I accord his opinion little weight.

Dr. Zaldivar also stated that since there was no evidence of coal workers' pneumoconiosis, pneumoconiosis did not contribute to Claimant's airway damage. "[A]medical diagnosis finding no coal workers' pneumoconiosis is not equivalent to a legal finding of no pneumoconiosis." *Hobbs v. Clinchfield Coal Co*., 45 F.3d 819, 821-22 (4th Cir. 1995). Dr. Crisalli opined that emphysema related to coal dust is a microscopic diagnosis and would not manifest itself with these types of pulmonary functions. He also stated that emphysema detectable by pulmonary function exam or bullous disease is always unrelated to coal mine dust.

[2] Dr. Crisalli correctly noted that a study from which Dr. Cohen drew the conclusion that asthma can be "fully" reversible states that obstructive lung disease is "often reversible." (EX 21 at 44; attachments to Nov. 8, 2004 letter from Employer to the undersigned.) I have acknowledged that asthma needs to not be completely reversible in making my determinations.

These conclusions defy the statutory definition of legal pneumoconiosis and undermine the credibility of the doctors' testimony. § 718.201.

Conclusion Under Compton

Therefore, I find that the physician opinion evidence also establishes the existence of legal pneumoconiosis. Further, weighing the x-ray evidence, the CT scan evidence, and the medical reports together at § 718.202(a) as required, I find that Claimant has clinical as well as legal pneumoconiosis. *Island Creek Coal Co. v. Compton*, 211 F.3d 203 (4th Cir. 2000)

Causation of Disability

I adhere to my finding of total disability at 718.204(b) as it was affirmed by the Board. The Board vacated my finding at § 718.204(c), instructing that if the issue of disability causation is reached on remand, I must consider all the relevant evidence regarding whether Claimant's total respiratory disability is due to pneumoconiosis, in light of the applicable law in the Fourth Circuit.

A miner shall be considered totally disabled due to pneumoconiosis if pneumoconiosis is a substantially contributing cause of his totally disabling respiratory or pulmonary impairment. Pneumoconiosis is a "substantially contributing cause" of the miner's totally disability if it: (i) has a material adverse effect on his respiratory or pulmonary impairment; or (ii) materially worsens a totally disabling respiratory or pulmonary impairment which is caused by a disease or exposure unrelated to coal mine employment. § 718.204(c)(1).

In *Toler v. Eastern Associated Coal Corp.*, 43 F.3d 109 (4th Cir. 1995), the court held that a physician's opinion regarding disability causation carries little weight if the physician diagnosed pneumoconiosis contrary to the ALJ's finding of the disease. However, the court circumscribed this holding in *Hobbs, supra.* p. 16, in which the court stated that "a medical diagnosis finding no coal workers' pneumoconiosis is not equivalent to a legal finding of no pneumoconiosis." *Id*. at 821-22.

The court held the medical opinions of physicians who concluded that the miner did not suffer from coal workers' pneumoconiosis did not necessarily conflict with the ALJ's conclusion that the miner suffered from pneumoconiosis, which encapsulates both the clinical and legal definition of pneumoconiosis. *See also Dehue Coal Co. v. Ballard*, 65 F.3d 1189 (4th Cir. 1995) (Physicians concluded that the miner did not suffer from coal workers' pneumoconiosis and that smoking-induced lung cancer caused the miner's pulmonary impairment. The court determined that these conclusions could properly be considered; they were not contrary to the ALJ's finding that the miner suffered from simple pneumoconiosis within the meaning of § 718.201 because coal workers' pneumoconiosis is only one of the many ailments that would satisfy the legal definition of pneumoconiosis.).

In *Scott v. Mason Coal Co*., 289 F.3d 263 (4th Cir. 2002), the court held that an ALJ who finds that a claimant suffers from pneumoconiosis and suffers from total disability may not credit

a medical opinion that the former did not cause the latter unless the ALJ identifies specific and persuasive reasons for so doing.

Dr. Crisalli found that the miner was unable to perform moderate to heavy work for a sustained period before bronchodilator treatment, but that the extent of his disability, unrelated to coal dust, was undetermined because it was unknown whether he would respond to bronchodilator treatment. Dr. Zaldivar found that Claimant was totally disabled due to asthma. Dr. Porterfield found that the miner was twenty-six percent disabled, stating that Claimant might be able to perform his last coal mine employment with frequent rest. Neither Dr. Zaldivar, Dr. Porterfield, nor Dr. Crisalli diagnosed legal pneumoconiosis in direct contradiction to my findings. Thus, in accordance with the aforecited case law, their opinions can carry only minimal weight.

Drs. Koenig and Cohen each opined that coal dust exposure was a significant contributing factor to the miner's pulmonary impairment. Neither Dr. Cohen nor Dr. Koenig determined that Claimant had clinical pneumoconiosis. However, in accordance with *Hobbs* and *Dehue*, the fact that neither physician found clinical pneumoconiosis does not necessarily conflict with my conclusion, and their conclusions, that Claimant suffered from legal pneumoconiosis in the form of COPD. Thus, their opinions regarding the etiology of Claimant's disability must be considered.

Drs. Koenig and Cohen rendered comprehensive assessments of Claimant's history of smoking, employment, symptomatology, and diagnostic testing. Each physician also incorporated medical literature in his opinion in support of his conclusion. Based on the foregoing, I find that Claimant is totally disabled due to pneumoconiosis.

The evidence establishes all of the elements of entitlement. Benefits will be awarded as of September 1, 2001, the first day of the month in which the claim was filed because the evidence does not establish the month of onset of total disability. § 725.503(b). Claimant's counsel has thirty days to file a fully supported fee application and his attention is directed to §§ 725.365 and 725.366. Employer's counsel has twenty days to respond with objections.

ORDER

IT IS ORDERED THAT Consolidation Coal Company:

I. Pay Claimant all the benefits to which he is entitled, augmented by one dependent, beginning as of September 1, 2001;

II. Pay Claimant all medical benefits to which he is entitled beginning as of September 1, 2001;

III. Reimburse the Black Lung Disability Trust Fund for interim payments made to Claimant; and

IV. Pay interest to the Black Lung Disability Trust Fund on unpaid benefits at the rates set forth in § 725.608.

A

DANIEL L. LELAND
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).